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CLINICAL REVIEW

Drowning

La noyade

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KEYWORD

Drowning

Abstract Drowning is responsible for significant preventable morbidity and mortality worldwide causing more deaths than war. Over the past 10 years our understanding of the causative factors, pathophysiology, management, outcomes, and even the terminology used has changed. Aggressive pulmonary support has proven to be essential to optimizing the victim's chances for a favorable outcome. Hypoventilation, steroids, dehydration, barbiturate coma, and neuromuscular blockade have not been shown to affect outcomes as previously thought. Further, no prognostic scale or clinical presentation has been found which accurately predicts long-term neurologic outcome. Simple preventative measures and educational efforts can be effective in preventing a major cause of morbidity and mortality in often young and healthy members of society.

Abstract La noyade est responsible de morbidité et mortalité significative évitable mondialement, causant plus de morts que la guerre. Durant les dix années précédentes notre compréhension des facteurs causatifs, de la physiopathologie, de la gestion, des aboutissements, et même de la terminologie a changée. L'assistance pulmonaire agressive s'est éprouvée être essentielle pour optimiser

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la probabilité d'un résultat favorable de la victime. L'hypoventilation, les stéroïdes, la déshydratation, le coma barbiturique, et le blocage neuromusculaire ne se sont pas montrés affecter des aboutissements comme on avait cru anciennement. En outre, il n'y a pas encore été trouvé ni d'échelle prognostique ni de présentation clinique qui prédit avec précision des conséquences neurologiques à longue durée. Des mesures préventives élémentaires et des efforts éducatifs peuvent être effectifs de prévenir une cause majeure de morbidité et mortalité parmi des membres de la société souvent jeune et en bonne santé.

Contents

What's new:

- Drowning is second only to motor vehicle collisions as the leading cause of injury mortality in South African children less than 15 years of age.
- Drowning is the leading cause of injury mortality in rural areas of Africa.
- Hyperventilation, steroids, dehydration, barbiturate coma, and neuromuscular blockade have not been consistently shown to improve outcomes after drowning.
- Aggressive pulmonary support is key to resuscitation after drowning.

African relevance:

- Drowning has been shown to be a leading cause of injury mortality in several countries in Africa with rates much higher than are seen in the United States and other well developed countries.
- Young children who drown in large household buckets are a major public health issue in poor communities throughout Africa.

Epidemiology

More people die from drowning each year than due to war. With an estimated 500,000 deaths worldwide due to drowning annually, submersion injury is a leading cause of preventable death.[1] Among children aged under 15 years, drowning is second only to motor vehicle crash as the leading cause of injury mortality in South Africa.[2–5] In a study of injury patterns in Uganda drowning was the leading cause of death in rural areas.[6] Toddlers and older teenagers are at greatest risk of death by drowning, with annual incidences of 2.45 and 1.47 per 100,000 respectively seen in the United States.[2] A study of drowning deaths in South Africa indicated a death rate of 12 per 100,000 for all ages in 2004.[7] Males account for almost 80% of victims older than 1 year.

The incidence of nonfatal submersion incidents is less well known. The Centers for Disease Control and Prevention estimates that for every child who dies by drowning in the United States, five receive emergency department care for a submersion event, and half of these children require hospitalization.[8] Among all age groups, it is estimated that one to four hospitalizations secondary to nonfatal submersions occur for every drowning fatality.[9–11]

Submersion injuries occur in domestic settings such as swimming pools, bathtubs, and large buckets as well as in natural bodies of water.[12] A South African study of 107 patients with "near-drowning" between 1976 and 1987 showed a racial predominance of incidents involving white victims occurring in the sea or swimming pools while incidents involving black or colored victims were more common to involve large household buckets.[5] Most submersion injuries in the South Africa and the United States occur on weekends between the hours of noon and 8:00 p.m.[5,9]

Definitions

The terminology used to describe submersion injuries has historically been confusing and impractical. Until 2005, *drowning* referred to death within 24 h of suffocation from submersion in

a liquid, whereas *near-drowning* described victims who survived at least 24 h past the initial event regardless of the outcome. In 2005, the World Health Organization (WHO) published a new policy defining drowning in an attempt to clarify documentation and better track submersion injuries worldwide. Drowning was redefined as "the process of experiencing respiratory impairment from submersion/immersion in liquid." The WHO policy further states that "drowning outcomes should be classified as: death, morbidity, and no morbidity... use of the terms wet, dry, active, passive, silent, and secondary drowning should no longer be used." [1] As such, the term *near-drowning* should not be used and the association of the term drowning with a fatal outcome should be abandoned.

Risk factors

Ethanol consumption in proximity with water is a major risk factor for submersion injury or death. Acute ethanol intoxication may be a contributing factor in 30–50% of drownings among adults and adolescents.[4,9] In one study of boating fatalities, most of which were due to drowning, an association between blood ethanol concentration and risk of death from drowning while using watercraft was established. Odds ratios of fatality from drowning followed a trend from 2.8 (95% CI 1.6, 4.8) for a BAC 1–49 mg/dL to 37.4 (95% CI 16.8, 83.0) for a BAC of 150 mg/dL or greater compared with sober case controls.[13]

The relationship between swimming ability and the risk of drowning is unclear. No direct evidence exists to suggest that inexperienced swimmers are more likely to drown. On the contrary, skilled swimmers have greater exposure to water and may be more prone to submersion incidents.[14]

Numerous medical conditions confer an increased likelihood of drowning or submersion injury. Seizure disorders sharply increase the chance of drowning among children and adolescents, and autism and other developmental and behavioral disorders increase risk in children.[15–17] Prolonged Q-T syndrome also is a risk factor for drowning. Laboratory studies show that immersion in cold water extends the Q-T interval. Interrogation of the automated internal cardiac defibrillator of a 12-year-old girl with prolonged Q-T syndrome who had a cardiac arrest on diving into the ocean revealed immediate further prolongation of the Q-T interval followed by a premature ventricular complex and subsequent ventricular tachycardia within 5 s.[18,19]

Pathophysiology

Unexpected submersion triggers breathe holding, panic, and a struggle to surface. Air hunger and hypoxia develop, and the victim begins to swallow water. As breath holding is overcome, involuntary gasps result in aspiration. The quantity of fluid aspirated rather than the composition determines subsequent pulmonary derangement. The historical emphasis on pathophysiologic differences between freshwater and saltwater aspiration with respect to resultant electrolyte imbalance, hemolysis, and fluid compartment shifting originated from animal studies conducted in the early 20th century and has been contradicted by more recent human research.

Significant intravascular abnormalities do not occur until the amount of aspirated water exceeds 11 mL/kg of body weight, and autopsy studies show that most drowning victims aspirate less than 4 mL/kg.[20] In one review of the hospital treatment of 91 submersion victims, no patient required emergent intervention for a significant electrolyte abnormality.[21] Aspiration of 1–3 mL/kg of either freshwater or saltwater destroys the integrity of pulmonary surfactant, leading to alveolar collapse, atelectasis, noncardiogenic pulmonary edema, intrapulmonary shunting, and ventilation-perfusion mismatch.[3] Profound hypoxia and metabolic and respiratory acidosis ensue, leading to cardiovascular collapse, neuronal injury, and, ultimately, death.

The classic hypothesis was that 10–15% of drowning victims die without aspirating a significant amount of water. Death from such "dry" drowning putatively results from severe laryngospasm causing hypoxia, convulsion, and death without fluid entering the lungs. An exhaustive review of the literature fails to corroborate this hypothesis.[22] So called dry drownings more appropriately reflect deaths from causes other than simple submersion.

Many factors may influence the pathophysiologic sequence of events in submersion injury and affect the chance of survival, including age, water temperature, duration and degree of hypothermia, the diving reflex, and the effectiveness of resuscitative efforts. Because of a lower ratio of body mass to surface area, children develop hypothermia more quickly and to a greater degree after immersion in cold water than adults. Hypothermia lowers cerebral metabolic rate and is neuroprotective to some extent for victims of submersion injury.[23] Despite dramatic case reports of patients surviving prolonged submersion in cold water with full neurologic recovery, however, in general hypothermia is a poor prognostic finding. Cold water immersion speeds the development of exhaustion, altered consciousness, and cardiac dysrhythmia. The *diving reflex* also may play a protective role in infant and child submersions. Activation of the diving reflex by fear or immersion of the face in cold water shunts blood centrally to the heart and brain. Apnea and bradycardia ensue, prolonging the duration of submersion tolerated without central nervous system damage.[24]

Symptoms and signs

Many submersion injuries are witnessed. Toddler drownings are an important exception, however, often occurring because of a lapse in supervision. Occasionally the history of coughing, choking, or vomiting in a patient found near a body of water suggests the diagnosis. Signs of *pulmonary injury* may be obvious in a submersion victim who is hypoxic, cyanotic, and in obvious respiratory distress or arrest. More subtle clues, such as increased respiratory rate and audible rhonchi, rales, or wheezes, should alert the clinician to evolving respiratory compromise. Submersion victims swallow a significantly greater volume of water than is aspirated, and gastric distension from positive-pressure ventilation during rescue is common. As a result, 60% of patients vomit after a submersion event.[3] Aspiration of gastric contents greatly compounds the degree of pulmonary injury and increases the likelihood that acute respiratory distress syndrome will ensue. Additionally, aspiration of particulate contaminants such as mud, sewage, and bacteria may obstruct the smaller bronchi and bronchioles and greatly increase the risk of infection both bacterial and fungal in nature.[25]

Victims with *central nervous system injury* may present with symptoms ranging from mild lethargy to coma with fixed and dilated pupils. Adverse neurologic findings on initial presentation do not preclude full neurologic recovery, although in general patients whose duration of submersion or resuscitation exceeds 25 min have an unfavorable outcome.[26] Central nervous system injury results from the initial hypoxic or ischemic insult and from the cascade of reperfusion injury that follows re-establishment of cerebral blood flow after an arrest. The release of inflammatory mediators and the generation of oxygen free radicals in the postresuscitative period contribute to cytotoxic cerebral edema, compromise of the blood–brain barrier, and increased intracranial pressure. Cerebral arteriolar vasospasm and enhanced platelet aggregation impede cerebral perfusion at the macrocirculatory and microcirculatory levels.[24]

Cardiac dysrhythmias may incite a submersion injury or develop as its consequence. Hypoxemia, acidosis, and, potentially, hypothermia are the primary factors responsible for dysrhythmias ranging from ventricular tachycardia and fibrillation to bradycardia-asystole. Electrolyte disturbances are rarely significant enough to be dysrhythmogenic.[21]

Other clinical sequelae of submersion injury may include acute *renal impairment,* present in approximately 50% of patients as the result of lactic acidosis; prolonged hypoperfusion; and, in some instances, rhabdomyolysis.[27] *Coagulopathy* as a consequence of associated hypothermia or disseminated intravascular coagulation may also occur.

Prognostic factors

Many factors may help predict patients who will survive a submersion injury neurologically intact. Submersion victims who arrive in the emergency department alert with normal hemodynamics are unlikely to experience neurologic impairment. Circumstantial variables that portend a poor outcome include victim age younger than 3 years, submersion for longer than 5 min, and initiation of cardiopulmonary resuscitation (CPR) more than 10 min after rescue.[3,28] With the exception of victim age, however, such measurements are generally either unknown or inaccurately estimated at the time of a patient's arrival in the emergency department. Objective findings on emergency department arrival that are associated with an unfavorable prognosis include hypothermia, severe acidosis, unreactive pupils, a Glasgow Coma Scale score of 3, and asystole or the need for ongoing CPR.[3,29–32] Neurologically intact survival is reported for individual patients even with several of these factors present, and none of several proposed scoring systems using combinations of these variables shows 100% predictive power.[28,30,33,34]

Differential considerations

The precipitants of a submersion injury, such as drug or ethanol intoxication, cardiac arrest, hypoglycemia, seizure, and attempted suicide or homicide, should be considered in a patient who is found unresponsive in the water. For pediatric victims, child abuse or neglect also must be considered as a potential etiology.

Potential head or cervical spine injury is an important consideration when a history of trauma is associated with the submersion. A review of 2244 cases of submersion injury in King, Pierce, and Snohomish counties in Washington State, however, identified only 11 (0.5%) patients with a cervical spine injury. Each patient had either clinical signs of serious trauma or a history of motor vehicle crash, fall from height, or diving into the water.[35] Unless such factors are present, routine cervical spine immobilization for submersion victims is not warranted.

Diagnostic studies

Cardiac monitoring and an electrocardiogram must be obtained to determine the presence of significant dysrhythmias or Q-T prolongation. Pulse oximetry, capnography, and arterial blood gases should be monitored closely in all submersion victims for signs of hypoxemia, hypercarbia, and acidosis. Blood glucose, serum creatinine, and electrolytes should be obtained, although serum creatinine and electrolytes are usually normal on initial presentation. Similarly, complete blood count is often normal with the exception of leukocytosis. Toxicologic screening may be appropriate depending on the circumstances of the submersion. Subsequently, evidence of renal failure, hepatic dysfunction, and disseminated intravascular coagulation may be noted on laboratory testing.

The initial chest radiograph may underestimate the severity of pulmonary injury, although infiltrates or pulmonary edema may be evident within hours. Cranial computed tomography is rarely contributory initially unless significant trauma or other pertinent pathology is suspected. Magnetic resonance imaging of the brain may predict neurologic outcome after submersion injury, but its prognostic value is not optimal until 3 or 4 days elapse.[34,36]

Management

Salient details of the events surrounding the accident should be ascertained rapidly. Resuscitation of pulseless and apnoeic patients should be attempted initially in most cases because bystander estimates of total submersion time are often inaccurate. The clinical presentation of severe hypothermia often mimics death, and case reports exist of functional recovery for individuals submerged for 66 min.[37,38]

For a victim without vital signs, outcome depends on the interval preceding CPR. Mouth-to-mouth assisted ventilation should begin immediately, even before the victim is extricated from the water. Chest compressions are impractical before extrication but should be initiated as soon as the individual is placed on a solid surface. Maneuvres such as those proposed by Heimlich and Patrick to remove fluid from the lungs are ineffective and dangerous in a victim at risk for aspiration and may delay ventilation. Use of such maneuvres is not recommended unless there is reason to suspect airway occlusion by a foreign body.[39]

On arrival in the emergency department, cardiac monitoring and continuous pulse oximetry should be established. A core temperature obtained with a low-reading probe is indicated for any unstable or lethargic patient. Rewarming a hypothermic patient may suffice for hemodynamic stabilization and improvement in mental status. Bedside blood glucose measurement should be performed. In a spontaneously breathing patient, monitoring for signs of developing pulmonary injury should be established. Initial chest radiographs are often unremarkable even in the setting of serious and evolving pathology.

The decision regarding tracheal intubation should be based on clinical impression and objective determination of the adequacy of oxygenation and ventilation. Apparent or developing respiratory distress, absence of protective airway reflexes, and significant associated head or chest injuries are indications. A $Paco_2$ greater than 50 mm Hg mandates intubation and mechanical ventilation. Patients unable to maintain an oxygen saturation greater than 90% or a Pao_2 greater than 60 mm Hg on high-flow oxygen require positive airway pressure to increase functional residual capacity, decrease intrapulmonary shunting, and reduce ventilation-perfusion mismatch. In an awake patient, this may be accomplished by face or nasal mask (continuous positive airway pressure), but the risk of potential gastric distension, vomiting, and aspiration must be considered. Otherwise, tracheal intubation and mechanical ventilation with positive end-expiratory pressure is necessary. The hemodynamic consequences of positive end-expiratory pressure must be monitored carefully because increased intrathoracic pressure may compromise venous return and cardiac output. Decreased cranial venous return may impede cerebral perfusion.

No consensus exists with regard to the appropriate length of resuscitative effort for hypothermic submersion victims in the emergency department. The safest parameter is to continue until the core temperature reaches at least 30–35 °C because cerebral death cannot be diagnosed accurately in hypothermic patients with temperatures below this level. This parameter may not always be practical, however, because brain-dead patients are often poikilothermic.

The administration of corticosteroids in the setting of submersion injury and potential acute respiratory distress syndrome does not improve outcome.[3,34] Similarly, empirical antibiotics do not increase survival and should be administered only to the rare patient who was submerged in grossly contaminated water or who shows signs of infection or sepsis.[3]

Interventions such as induced or permissive hypothermia aimed at attenuating reperfusion injury after anoxic brain insult are the focus of intense investigative effort, but at present no consensus exists regarding their use in submersion injury. A case report of twin toddlers with identical submersion injury and subsequent prolonged cardiac arrest indicates that therapeutic hypothermia may be a factor in influencing a good neurologic outcome. One twin was treated with therapeutic hypothermia for 72 h and had a return to normal neurologic status. The other twin was not cooled and survived, but with significant neurologic impairment.[40] Reports such as this and studies in the resuscitation literature indicate an emerging role for therapeutic hypothermia in some drowning victims. Barbiturate-induced coma, aggressive diuresis, neuromuscular blockade, and hyperventilation do not improve neurologic outcome and, particularly in the case of hyperventilation, may be harmful.[24]

Disposition

Symptomatic patients must be admitted for treatment. Patients with a history of apnea, unconsciousness, or hypoxia and any patients who manifest dysrhythmia or an abnormal chest radiograph also require admission. Patients who are asymptomatic on presentation to the emergency department, maintain a normal room air oxygen saturation, and have no chest radiograph or arterial blood gas abnormalities can be discharged safely after an observation period of 6 h.[34,41] Careful instructions regarding symptoms or signs of delayed pulmonary complications are necessary, and the patient should be discharged in the care of a competent relative or friend.

Preventive efforts

Public awareness of preventive measures and an emphasis on public education with regard to CPR and the dangers of ethanol use in conjunction with water-related activities have contributed significantly to a reduction in drowning fatalities in the United States. A longitudinal study of drownings over a 21-year period in King County, Washington, notes that the incidence of death attributable to ethanol use has decreased by 81%.[3]

Parental education regarding the danger of pediatric drowning is an important focus of preventive effort. Inadequate supervision of children playing in or near water is one of the most common causes of pediatric submersion death, underscoring the importance of increasing awareness of the need for constant oversight of children in this setting.[11,41] Most pediatric submersion injuries in swimming pools occur at the victim's home.[11] In most cases, the child was last seen in the house, was left unattended for a moment, and entered the pool on an unfenced side closest to the home with no audible splash or screaming.[11] Adequate and fully circumferential fencing of residential pools is a current recommendation of the American Academy of Pediatrics. A meta-analysis of the literature regarding the efficacy of this preventive step reports an odds ratio of 0.27 for drowning or submersion in a properly fenced compared with a non-fenced pool.[41,43] Unfortunately, legislation requiring appropriate fencing is poorly adhered to with only 40% of households compliant in one study.[44]

Education directed at preventing household bucket deaths may be a high yield health prevention effort in communities where this is prevalent.

Pool covers are inadequate and potentially dangerous as barriers. Solar blankets do not support the weight of a child and can enmesh and obscure a struggling victim from view. A rigid pool cover may convey a false sense of stability to a child tempted to walk across its surface and is considered an insufficient substitute for effective four-sided fencing.[42]

Medical care providers are a vital resource for enhancing public awareness of the importance of these measures. The literature supports the concept that education in the emergency department with regard to drowning prevention can have a positive impact on patient and family awareness of steps to lessen the likelihood of catastrophic drowning or submersion injury.[45,46]

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